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Original Article

Phenotypic Detection of Carbapenamases, Amp C β-Lactamese and Their Co-Existence Among Esbl Producing E. Coli and Klebsiella Pneumoniae

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ABSTRACT

Background: *Escherichia coli* and *Klebsiella pneumoniae* are major Gram-negative pathogens with increasing β-lactamase-mediated resistance. This study evaluated antimicrobial susceptibility and phenotypic detection of ESBL, AmpC β-lactamase, carbapenemase, and metallo-β-lactamase production among clinical isolates.
Methods: A cross-sectional study was conducted in the Department of Microbiology, P.D.U. Medical College and Hospital, Rajkot, from January to December 2025. Non-duplicate clinical isolates of *E. coli* and *K. pneumoniae* were identified by standard microbiological methods. Antimicrobial susceptibility testing was performed by Kirby–Bauer disk diffusion. ESBL, AmpC, carbapenemase, and metallo-β-lactamase production were detected phenotypically using combined disk methods, cefoxitin–cloxacillin testing, mCIM, and eCIM.
Results: Of 2035 isolates, 845 (41.5%) were *E. coli* and 1190 (58.5%) were *K. pneumoniae*. *E. coli* was isolated mainly from urine, whereas *K. pneumoniae* predominated in pus/swab specimens. Confirmed ESBL production was detected in 306 (15.0%) isolates and was higher in *E. coli* than *K. pneumoniae* (22.0% vs 10.1%; $p<0.001$). Among ESBL producers, AmpC co-production occurred in 44 (14.4%), mCIM positivity in 8 (2.6%), and eCIM positivity in 1 (0.3%). Carbapenem susceptibility was higher in *E. coli* than *K. pneumoniae*, while all isolates were susceptible to colistin.
Conclusion: Species-specific resistance patterns and β-lactamase co-production emphasize the need for routine phenotypic detection and local antimicrobial stewardship.

Keywords*: Escherichia coli; Klebsiella pneumoniae; ESBL; AmpC β-lactamase; carbapenemase.*

INTRODUCTION

Antimicrobial resistance (AMR) has become one of the biggest challenges in the current era of medicine, making the common antibiotic less effective and causing increased morbidity, mortality, length of stay in the hospital and costs. Global estimates revealed that the bacterial AMR caused millions of deaths with significant contribution of Gram-negative pathogens [1]. Hence, WHO has declared the antibiotic-resistant Gram-negative bacteria, including the carbapenem-resistant Enterobacterales, as pathogens of urgent priority for surveillance, prevention and treatment [2].

Of the various Gram-negative bacteria, E. coli and K. pneumoniae are very significant due to their common causative role in urinary tract infection, wound infection, bloodstream infection, respiratory infection and hospital-acquired infection. They gain significance because of their tendency of acquiring and disseminating resistance genes through plasmids and other mobile genetic elements. β-lactam antibiotics are one of the most common drugs used for treating Gram-negative infections but are increasingly becoming ineffective due to various β-lactamases hydrolysing the β-lactam ring [3].

The extended-spectrum β-lactamases (ESBLs) are one of the most common resistance mechanisms in E. coli and K. pneumoniae. ESBLs produce isolates hydrolyse extended-spectrum cephalosporins and aztreonam, and are commonly associated with resistance to aminoglycosides, fluoroquinolones and cotrimoxazole [4]. The AmpC β-lactamases cause problems because they make isolates resistant to many penicillins, cephalosporins, cephamycins and β-lactam/β-lactamase inhibitor combinations [5]. The carbapenemases (including metallo-β-lactamases such as NDM, VIM and IMP enzymes) are of major concern because they make the carbapenems ineffective and are generally saved for use against ESBL/AmpC producing organisms [6].

In India, the issue of β-lactamase-mediated resistance assumes importance since the tertiary care hospitals see a large number of infections and hence experience strong selection pressure from the use of antibiotics. Multi-centric Indian data has highlighted the regional differences in ESBL prevalence and molecular patterns in E. coli and K. pneumoniae, stressing the need for regional surveillance and not just national or international estimates [7]. It is essential to know the local patterns of antimicrobial susceptibility for formulating local policies on empirical therapy, infection control and antimicrobial stewardship, especially since the patterns vary by organism, site of isolation, population and healthcare facility.

Hence, the detection of β-lactamase production in the laboratory is an integral part of the routine practice in microbiology. Phenotypic tests like the combined disk test for ESBL, the cefoxitin based method for AmpC and the carbapenem inactivation method are useful and inexpensive means of detecting clinically significant resistance mechanism in resource limited settings. The modified carbapenem inactivation method (mCIM) has been found to be a good phenotypic method of detecting carbapenemases in Enterobacterales [8], whereas the EDTA modified carbapenem inactivation method (eCIM) is a good method to distinguish metallo-β-lactamases among mCIM positive isolates [9].

This study has thus been conducted to determine the antimicrobial susceptibility profile and phenotypic distribution of ESBL, AmpC β-lactamase, carbapenemase and metallo-β-lactamase production (and co-production patterns) among E. coli and K. pneumoniae isolated from a tertiary care hospital in Rajkot, Gujarat.

MATERIALS AND METHODS

Study design and setting

This cross-sectional study was conducted in the Department of Microbiology, P.D.U. Medical College and Hospital, Rajkot, Gujarat, India, from January 2025 to December 2025. The study evaluated the antimicrobial susceptibility profile and phenotypic detection of extended-spectrum β-lactamase (ESBL), AmpC β-lactamase, carbapenemase, and their co-production among clinical isolates of *Escherichia coli* and *Klebsiella pneumoniae*.

The calculated minimum sample size was 250 isolates; however, all eligible isolates recovered during the study period were included in the final analysis, yielding 2035 isolates.

Study isolates and eligibility criteria

All non-duplicate clinical isolates of *E. coli* and *K. pneumoniae* obtained from samples received for routine bacterial culture and antimicrobial susceptibility testing were included. Isolates were recovered from urine, pus/wound swabs, blood, body fluids, sputum/endotracheal aspirates, and other relevant clinical specimens from patients of all age groups and both sexes, including outpatient and inpatient departments.

Isolates other than *E. coli* and *K. pneumoniae* were excluded. Repeat isolates from the same patient and same specimen type were excluded to avoid duplication.

Sample processing and bacterial identification

Clinical specimens were processed using standard microbiological techniques. Direct Gram staining was performed where appropriate. Samples were inoculated onto routinely used culture media, including blood agar, MacConkey agar, nutrient agar, and chocolate agar as applicable, followed by aerobic incubation at 37°C for 18–24 hours.

Bacterial isolates were identified based on colony morphology, Gram staining, motility, and standard biochemical reactions, including catalase, oxidase, indole, citrate utilization, urease, triple sugar iron agar reaction, methyl red test, phenylalanine deaminase test, and sugar fermentation tests. *E. coli* was identified by characteristic lactose-fermenting colonies, motility, indole positivity, citrate negativity, and urease negativity. *K. pneumoniae* was identified by mucoid lactose-fermenting colonies, non-motility, indole negativity, citrate positivity, and urease positivity.

Antimicrobial susceptibility testing

Antimicrobial susceptibility testing was performed by the modified Kirby–Bauer disk diffusion method on Mueller–Hinton agar. The bacterial inoculum was adjusted to 0.5 McFarland turbidity standard before inoculation. Antibiotic disks

were applied according to standard spacing recommendations, and plates were incubated aerobically at 35–37°C for 16–18 hours.

Zone diameters were measured in millimeters and interpreted according to Clinical and Laboratory Standards Institute guidelines. Antibiotics tested included β-lactam/β-lactamase inhibitor combinations, cephalosporins, carbapenems, aminoglycosides, fluoroquinolones, tetracyclines, cotrimoxazole, and urine-specific agents where applicable. Nitrofurantoin, norfloxacin, cefazolin, and fosfomycin results were analyzed only for urinary isolates.

Colistin susceptibility was assessed using colistin screen agar with graded colistin concentrations. The lowest concentration completely inhibiting visible growth was recorded as the minimum inhibitory concentration, and results were interpreted using accepted clinical breakpoints.

Phenotypic detection of ESBL production
All isolates were screened for possible ESBL production using disk diffusion screening criteria for third-generation cephalosporins and aztreonam. Isolates with reduced susceptibility to cefotaxime, ceftazidime, ceftriaxone, cefpodoxime, or aztreonam were subjected to confirmatory ESBL testing.

ESBL production was confirmed by the combined disk diffusion method using ceftazidime and cefotaxime disks alone and in combination with clavulanic acid. An increase of ≥5 mm in the inhibition zone diameter around the cephalosporin–clavulanate disk compared with the corresponding cephalosporin disk alone was interpreted as ESBL positive.

Phenotypic detection of AmpC β-lactamase
Isolates were screened for AmpC β-lactamase production using cefoxitin disk diffusion and resistance to third-generation cephalosporins. Isolates with reduced cefoxitin susceptibility were further tested by the cefoxitin–cloxacillin combined disk method.

AmpC production was confirmed when the zone diameter around the cefoxitin–cloxacillin disk increased by ≥5 mm compared with cefoxitin alone. Inducible AmpC production was also assessed by disk antagonism, where blunting of the inhibition zone of a substrate disk toward an inducer disk was considered positive.

Phenotypic detection of carbapenemase and metallo-β-lactamase
Isolates showing intermediate susceptibility or resistance to one or more carbapenems were subjected to carbapenemase testing. Carbapenemase production was detected using the modified carbapenem inactivation method (mCIM). Metallo-β-lactamase production was assessed using the EDTA-modified carbapenem inactivation method (eCIM).

An mCIM-positive result was interpreted as phenotypic evidence of carbapenemase production. Among mCIM-positive isolates, an eCIM-positive result indicated metallo-β-lactamase production.

Definition of β-lactamase co-production
Confirmed ESBL-producing isolates were further evaluated for co-production of AmpC β-lactamase and carbapenemase. Co-production phenotypes were classified as:

- ESBL with AmpC β-lactamase production;
- ESBL with carbapenemase production, based on mCIM positivity;
- ESBL with metallo-β-lactamase production, based on eCIM positivity.

For co-production analyses, percentages were calculated using confirmed ESBL-producing isolates as the denominator for each species: *E. coli*, n = 186, and *K. pneumoniae*, n = 120.

Quality control
Quality control was performed using standard reference strains. *E. coli* ATCC 25922 was used as the negative control for ESBL testing and as the indicator strain for mCIM/eCIM testing. *K. pneumoniae* ATCC 700603 was used as the ESBL-positive control. *Pseudomonas aeruginosa* ATCC 27853 was used for colistin quality control.

Statistical analysis
Data were entered into Microsoft Excel and analyzed using appropriate statistical software. Categorical variables were summarized as frequency and percentage in the format *n (%)*. Comparisons between *E. coli* and *K. pneumoniae* were performed using the chi-square test or Fisher's exact test, as appropriate. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 2035 eligible isolates were analyzed, comprising 845 (41.5%) *Escherichia coli* and 1190 (58.5%) *Klebsiella pneumoniae*. The age distribution differed significantly by species (χ^2=47.31, df=6; p<0.001), and the >50-year age group contributed the largest proportion for both organisms. Isolates from male patients were more frequent among *K. pneumoniae* than among *E. coli* (667 [56.1%] vs 421 [49.8%]; χ^2=7.70, df=1; p=0.006).

The specimen-source distribution also differed significantly between species (χ^2=335.34, df=4; p<0.001). *E. coli* was isolated predominantly from urine specimens (462 [54.7%]), whereas *K. pneumoniae* was isolated most frequently from pus/swab specimens (564 [47.4%]). Overall, pus/swabs accounted for 864 (42.5%) isolates, followed by urine with 687 (33.8%) isolates (Table 1 and Figure 1).

Table 1. Demographic and specimen-source distribution by species.

Characteristic	E. coli (n=845), n (%)	K. pneumoniae (n=1190), n (%)	Test statistic	p value
Age group, years			**χ^2=47.31; df=6**	**<0.001**
<1	33 (3.9)	98 (8.2)		
1–10	93 (11.0)	59 (5.0)		
11–20	43 (5.1)	73 (6.1)		
21–30	136 (16.1)	180 (15.1)		
31–40	115 (13.6)	217 (18.2)		
41–50	132 (15.6)	170 (14.3)		
>50	293 (34.7)	393 (33.0)		
Sex			**χ^2=7.70; df=1**	**0.006**
Male	421 (49.8)	667 (56.1)		
Female	424 (50.2)	523 (43.9)		
Specimen source			**χ^2=335.34; df=4**	**<0.001**
Urine	462 (54.7)	225 (18.9)		
Blood	44 (5.2)	124 (10.4)		
Pus/swabs	300 (35.5)	564 (47.4)		
Sputum/ETA	8 (0.9)	132 (11.1)		
Body fluids	31 (3.7)	145 (12.2)		

Percentages are column percentages. Chi-square tests compare distributions between species.

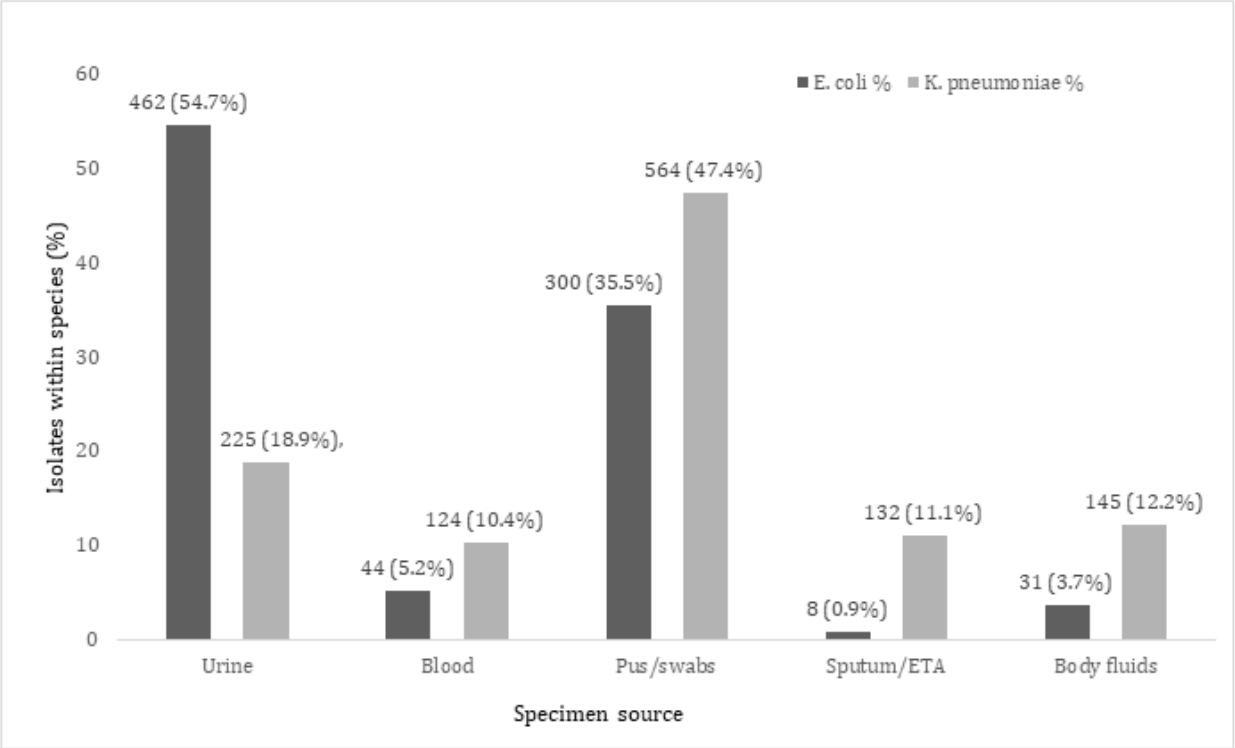

Figure 1. Distribution of specimen sources by bacterial species.

Bars show the percentage of isolates within each species.

Phenotypic detection of ESBL, AmpC β-lactamase and carbapenemase

Of the 2035 isolates, 1777 (87.3%) were screen-positive for possible ESBL production and 306 (15.0%) were confirmed ESBL producers. Confirmed ESBL production was significantly more frequent in *E. coli* than in *K. pneumoniae* (186 [22.0%] vs 120 [10.1%]; χ^2=55.03; p<0.001).

Among confirmed ESBL producers, AmpC co-production was identified in 44 (14.4%) isolates, with similar rates in *E. coli* and *K. pneumoniae* (27 [14.5%] vs 17 [14.2%]; χ^2=0.01; p=0.932). Carbapenemase production by mCIM was detected in 8 (2.6%) ESBL-positive isolates, and eCIM positivity consistent with metallo-β-lactamase production was detected in 1 (0.3%) isolate, which was *E. coli* (Table 2 and Figure 2).

Table 2. Phenotypic detection of β-lactamase production and co-production.

Phenotypic result	Denominator	E. coli n (%)	K. pneumoniae n (%)	Total n (%)	Test	p value
ESBL screen-positive	All isolates	719 (85.1)	1058 (88.9)	1777 (87.3)	χ^2=6.51	0.011
Confirmed ESBL	All isolates	186 (22.0)	120 (10.1)	306 (15.0)	χ^2=55.03	<0.001
AmpC screen-positive	Confirmed ESBL isolates	186 (100.0)	119 (99.2)	305 (99.7)	Fisher exact	0.392
Confirmed ESBL + AmpC	Confirmed ESBL isolates	27 (14.5)	17 (14.2)	44 (14.4)	χ^2=0.01	0.932
mCIM positive	Confirmed ESBL isolates	5 (2.7)	3 (2.5)	8 (2.6)	Fisher exact	1.000
eCIM positive (MBL)	Confirmed ESBL isolates	1 (0.5)	0 (0.0)	1 (0.3)	Fisher exact	1.000

Tests compare E. coli with K. pneumoniae. Fisher's exact test was used where expected cell counts were small. mCIM, modified carbapenem inactivation method; eCIM, EDTA carbapenem inactivation method; MBL, metallo-β-lactamase.

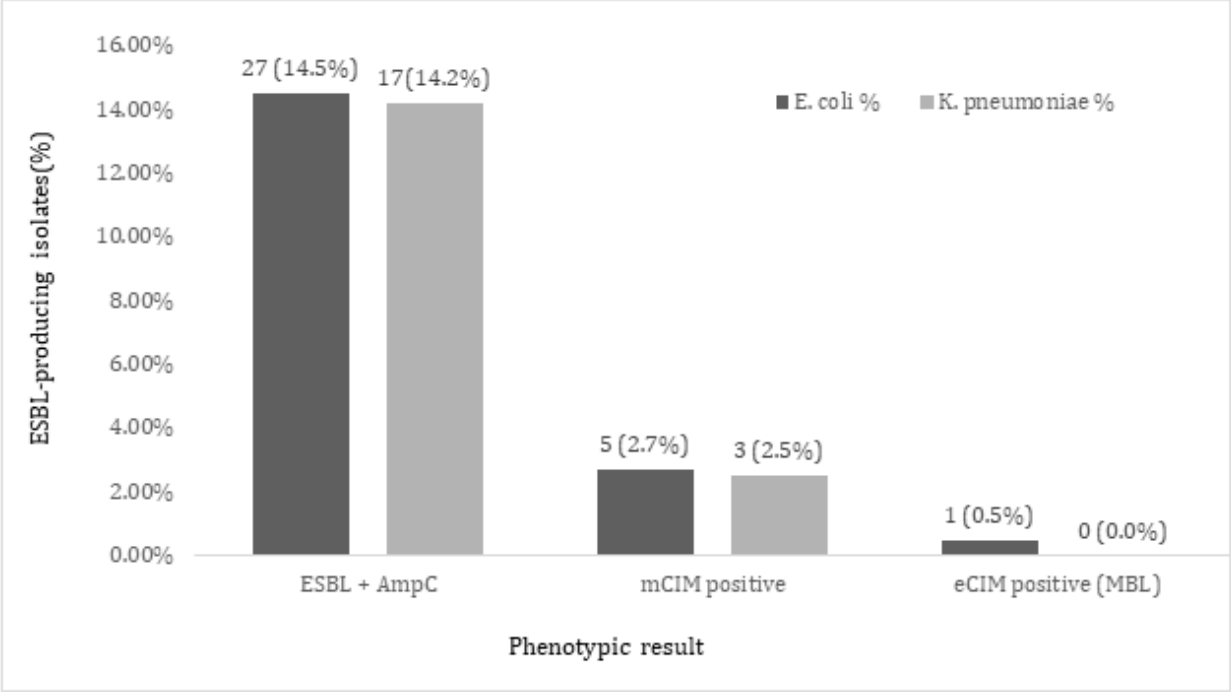

Figure 2. Co-production phenotypes among confirmed ESBL-producing isolates.

Percentages use confirmed ESBL producers as the denominator for each species: E. coli n=186; K. pneumoniae n=120.

Antimicrobial susceptibility profile

Both organisms showed complete in-vitro susceptibility to colistin. Compared with *K. pneumoniae*, *E. coli* demonstrated significantly higher susceptibility to piperacillin-tazobactam, carbapenems and aminoglycosides (Table 3). Carbapenem susceptibility remained higher in *E. coli* (86.2–86.5%) than in *K. pneumoniae* (74.6–75.2%). Third-generation cephalosporins and fluoroquinolones showed low activity in both species.

Among urinary isolates, fosfomycin was active against 423/462 (91.6%) *E. coli* isolates and nitrofurantoin against 374/462 (81.0%) *E. coli* isolates. Nitrofurantoin activity against urinary *K. pneumoniae* isolates was lower at 98/225 (43.6%).

Table 3. Selected antimicrobial susceptibility profile by species.

Antimicrobial agent	E. coli susceptible n (%)	K. pneumoniae susceptible n (%)	Test	p value
Piperacillin-tazobactam	435 (51.5)	317 (26.6)	χ^2=130.87	<0.001
Cefepime	259 (30.6)	254 (21.3)	χ^2=22.70	<0.001
Ceftazidime	163 (19.3)	146 (12.3)	χ^2=18.91	<0.001
Imipenem	728 (86.2)	888 (74.6)	χ^2=40.19	<0.001
Meropenem	731 (86.5)	895 (75.2)	χ^2=39.28	<0.001
Ertapenem	730 (86.4)	892 (75.0)	χ^2=39.93	<0.001
Amikacin	522 (61.8)	444 (37.3)	χ^2=118.60	<0.001
Gentamicin	510 (60.4)	456 (38.3)	χ^2=96.22	<0.001
Netilmicin	599 (70.9)	483 (40.6)	χ^2=182.19	<0.001
Tetracycline	360 (42.6)	567 (47.6)	χ^2=5.07	0.024
Doxycycline	430 (50.9)	683 (57.4)	χ^2=8.44	0.004
Ciprofloxacin	188 (22.2)	233 (19.6)	χ^2=2.14	0.143
Levofloxacin	195 (23.1)	253 (21.3)	χ^2=0.95	0.330
Cotrimoxazole	402 (47.6)	509 (42.8)	χ^2=4.61	0.032
Colistin	845 (100.0)	1190 (100.0)	Not estimable	—

For systemic agents, percentages use total species denominators. Tests compare susceptible versus non-susceptible isolates between species using chi-square tests. Colistin was not compared statistically because all isolates in both groups were susceptible.

DISCUSSION

The current study describes a substantial and contemporary data set on tertiary care hospital isolates of E. coli and K. pneumoniae in Western India, combining organism-wise antimicrobial susceptibility with phenotypic confirmation of ESBL, AmpC β-lactamase, carbapenemase, and metallo-β-lactamase co-production. The predominance of K. pneumoniae over E. coli in the isolate pool, combined with the different specimen composition (E. coli isolated predominantly from urine cultures and K. pneumoniae from pus/swab specimens), underlines the specific clinical ecology of these Enterobacterales within the same setting. Importantly, the empirical Gram-negative antibiogram approach may overlook clinically relevant species-wise resistance trends.

The confirmed ESBL rate in our study was 15.0%, which was significantly higher in E. coli compared to K. pneumoniae (22.0% vs 10.1%). The organism-wise pattern observed in our study is somewhat consistent with the multicenter study of Karnataka, conducted by Rao et al. [10]. The study evaluated 2000 isolates equally divided into E. coli and K. pneumoniae from 5 medical centers in Karnataka state. They reported an overall ESBL rate of 57.5% with higher ESBL production by E. coli than K. pneumoniae (61.4% vs 46.2%). They also detected 58 ESBL–AmpC co-producing isolates that were predominantly E. coli rather than K. pneumoniae and showed low imipenem resistance among ESBL producers. The lower ESBL rate in our study may be due to difference in the case mix, geographic location, denominator, non-duplicate isolation, and confirmatory criteria. Nonetheless, the consistent finding of greater ESBL rate in E. coli calls for a need of species-wise ESBL surveillance rather than pooled Enterobacterales ESBL reporting.

The ESBL rate in our study was lower than that by Kazemian et al. [11] evaluating hospitalized isolates of K. pneumoniae and E. coli. They found phenotypic ESBL production in 40.0% of K. pneumoniae and 35.4% of E. coli. Also, AmpC production in 20.0% of K. pneumoniae and 9.2% of E. coli, carbapenemase production using modified Hodge test in 43.3% of K. pneumoniae and 27.7% of E. coli, and concomitant carriage of ESBL, AmpC, and carbapenemase genes in K. pneumoniae isolates. The difference in the rate of mCIM positive isolates among ESBL producers may indicate lower carbapenemase selective pressure in our setting or methodological and epidemiological differences (hospitalized cases only vs outpatient and hospitalized vs molecular confirmation).

The specimen distribution in our study underlines the important role of E. coli in urinary infections but also highlights the wider spectrum of invasive and wound-associated infections caused by K. pneumoniae. For instance, the multicenter Indian community-acquired UTIs study by Mohapatra et al. [15] evaluated 2459 urine samples with the culture positivity rate of 10.1% and a female predominance. E. coli and K. pneumoniae accounted for 68.0% and 17.6% of uropathogens, respectively. The authors found ESBL production in 44.8% of isolates, carbapenem resistance in 4.3%, ESBL production in 52.8% of uropathogenic E. coli, and no fosfomycin resistance among uropathogenic E. coli. The current study extends

the above-mentioned findings to a broader specimen diversity, with the urinary E. coli remaining prominent but K. pneumoniae contributing considerably to pus/swab, respiratory, body fluid, and blood cultures.

The clinical importance of β-lactamase co-production is evidenced by the masking of phenotypic detection, reduced therapeutic options, and multidrug resistance. ESBL-AmpC co-production was detected in 14.4% of confirmed ESBL producers, with no significant difference between E. coli and K. pneumoniae isolates. This is different from the multicenter Karnataka study by Rao et al. [10], where ESBL-AmpC co-production was lower overall and more prevalent among E. coli than in K. pneumoniae. Another example is the study by Salvia et al. [12] analyzing 400 Enterobacteriaceae isolates and showing 304 (76.0%) to be multidrug resistant. In this MDR-enriched dataset, ESBL rate was 50.4% using phenotypic confirmatory disc diffusion test, AmpC rate 13.1% using boronic acid test, and MBL rate 14.8% using Carba NP test. The authors detected ESBL-AmpC co-production, ESBL-MBL co-production, AmpC-MBL co-production, and rare co-production of all β-lactamase types. The lower rate of MBL positive isolates in our study (1 eCIM positive isolate among confirmed ESBL producers) indicates a smaller MBL burden in our setting. Nevertheless, the occurrence of ESBL-AmpC co-production supports the need for testing for overlapping β-lactamase mechanisms routinely.

The above-mentioned information is also supported by the findings of Mirza et al. [13] evaluating community-acquired infections in India. They found that β-lactamase coexistence was not specific for the hospital-acquired or ICU setting. In their study, 417 multidrug-resistant community-acquired isolates were detected, 293 isolates were positive for at least one major β-lactamase mechanism, and the coexistence of mechanisms included ESBL-AmpC, ESBL-MBL, and AmpC-MBL combinations. This is particularly relevant to our findings, as the significant number of ESBL screen-positive isolates was detected prior to confirmatory testing, and a considerable portion of confirmed ESBL producers possessed AmpC phenotype. The above-mentioned facts indicate that the diagnostic algorithm of ESBL confirmation only may underestimate clinically relevant β-lactamase complexity.

The carbapenem findings in our study require particular consideration. Carbapenem susceptibility was still higher in E. coli than in K. pneumoniae but was only about three-fourth for imipenem, meropenem, and ertapenem in K. pneumoniae. Simultaneously, mCIM rate among confirmed ESBL producers was low, and eCIM rate was even lower. This could indicate that carbapenem non-susceptibility in some isolates was related to mechanisms other than carbapenemase production among ESBL producers, such as porin alteration, efflux, co-expression of AmpC or ESBL along with permeability defect, or carbapenemase production among non-ESBL confirmed isolates. The contrast with the much higher carbapenemase rate in the study by Kazemian et al. [11] and the low imipenem resistance rate in the study by Rao et al. [10] put our setting into the intermediate position: carbapenems remain useful, especially for E. coli, but the reduced susceptibility in K. pneumoniae requires caution.

The antimicrobial susceptibility profile has therapeutic implications as well. Third-generation cephalosporins and fluoroquinolones had low activity in both organisms, whereas aminoglycosides, piperacillin-tazobactam, and carbapenems were more active against E. coli than K. pneumoniae. Importantly, K. pneumoniae accounted for the majority of isolates in our setting. In the study of Amladi et al. [14] evaluating carbapenem-resistant E. coli and Klebsiella spp. causing UTIs, very high resistance to piperacillin-tazobactam, ciprofloxacin, amikacin, and gentamicin was observed among carbapenem-resistant isolates, and fosfomycin maintained activity against 98.9% of carbapenem-resistant E. coli and 94.0% of carbapenem-resistant Klebsiella spp. In addition, nitrofurantoin activity was lower than fosfomycin and NDM and OXA-48-like carbapenemase mechanisms were documented. Fosfomycin had high activity (91.6%) against urinary E. coli in our study, and nitrofurantoin activity against urinary E. coli was 81.0%, indicating their continued role as sparing agents of the urinary tract. But nitrofurantoin activity against K. pneumoniae was low, and it underlined the need for organism-specific reporting in UTIs.

Colistin susceptibility in both organisms was 100% in our study, which is promising but should be considered carefully. Amladi et al. [14] reported a lower colistin susceptibility among carbapenem-resistant UTI isolates, particularly among Klebsiella spp., and Shankar et al. [16] described 65 colistin-resistant carbapenem-resistant K. pneumoniae isolates from India with colistin MICs from 4 to 256 µg/mL. In that study, OXA-48-like carbapenemase genes were common, ESBL gene co-carriage was frequent, mgrB alteration was identified in a subset of isolates, and plasmid-mediated mcr genes were absent. The findings indicate that colistin resistance could be achieved through chromosomal mechanism and clonal expansion even in the absence of mcr. Thus, continuous monitoring and phenotypic confirmatory testing remains essential, especially for carbapenem-resistant K. pneumoniae.

Strengths of the study include large sample size, non-duplicate isolates, various specimens, and combination of susceptibility with ESBL, AmpC, carbapenemase, and metallo-β-lactamase phenotyping. Limitations include single-center design, absence of molecular confirmation, absence of clinical and antibiotic exposure data, absence of phenotypic colistin susceptibility testing, and limited co-production analysis.

So the current study demonstrates considerable β-lactamase-mediated resistance in E. coli and K. pneumoniae, with higher ESBL rate in E. coli, increased resistance in K. pneumoniae, considerable ESBL-AmpC co-production, and retained fosfomycin activity against urinary E. coli. The results call for routine phenotypic β-lactamase testing, organism-wise antibiograms, and continuous carbapenem and colistin surveillance.

CONCLUSION

This study demonstrates considerable β-lactamase-mediated resistance among E. coli and K. pneumoniae, with significant species-specific differences. Routine phenotypic β-lactamase detection, organism-specific antibiograms, and carbapenem and colistin surveillance are essential for empirical treatment and antimicrobial stewardship.

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